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Angiotensin converting enzyme inhibition prevents development of muscle and nerve dysfunction and stimulates angiogenesis in streptozotocin-diabetic rats.

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The effects of the angiotensin converting enzyme inhibitor lisinopril on slow and fast twitch muscle contractile properties, nerve conduction and hypoxic resistance, and muscle and nerve capillary density were examined in streptozotocin-diabetic rats. Prolongation of soleus contraction and relaxation were partially prevented by treatment (p less than 0.01). A 22% deficit in fast twitch extensor digitorum longus tetanic tension production was also ameliorated (p less than 0.01). Sciatic motor and sensory conduction velocity, 25% and 12% reduced by diabetes respectively, were 75% normalized by lisinopril (p less than 0.01). There was a 47% increase in resistance to hypoxic conduction block with diabetes (p less than 0.01). Lisinopril treatment resulted in normal hypoxic resistance. Capillarization of nerve and muscle was little affected by diabetes; however, there was a 17% increase in capillary density in sciatic nerve, and a 40% increase in extensor digitorum longus muscle with lisinopril (p less than 0.01). For soleus, a smaller treatment-induced increase in capillary density led to an elevated capillary/muscle fibre ratio (p less than 0.01). These results suggest that lisinopril promoted angiogenesis. It was concluded that the beneficial effect of preventive lisinopril treatment is likely to depend upon a reduction of peripheral vascular resistance and improvement of tissue blood flow, which implicates relative hypoxia as an important factor in the development of myopathy and neuropathy in experimental diabetes.

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